Skin and Soft Tissue Infections

Introduction

"Skin and soft tissue" infection is a way of saying any infection "from epidermis to bone." These infections are lumped together because they share common **infectious organisms**. Skin floras are always present on the outside of our skin and therefore the most likely to cause a soft tissue infection. Those organisms are ***Staph. aureus*** and ***Strep. pyogenes*** **(group A strep)**. It isn't that other organisms cannot cause soft tissue infections (the stage IV sacral decubitus can be infected with literally any organism), it's just that the classic presentations, the ones you need to learn at this stage of training, are with these organisms. Strep and staph for every infectious diagnosis in this lesson, unless we call out specific exceptions as we go.

We will be following two general principles to explain the infection's progression and behavior.

The first principle is, *"Strep spreads out, staph spreads down."* This is NOT used to correlate an organism with depth of invasion of the skin layers. Strep and staph can both be as deep as they want. This is used to correlate the disease progression. Erysipelas and nec fac are rapidly progressive, rapidly expanding infections that stay in the plane of infection, allowing a spread outward, covering more surface area. These infections are caused by strep. Strep spreads out. Abscesses and osteomyelitis are not rapidly progressive and represent a depth of invasion in the horizontal plane of the skin, going down, going deep. These are caused by staph. Staph goes down.

The second principle is the depth of infection to name the diagnosis. This is only a mental exercise used to organize the lesson. Most of these diagnoses never need a biopsy, so you will never literally see the depth of infection. But this lesson is organized from the most superficial to the most deep, with abscess as special asterisk at the end. This visualization (Fig. 4.1) is a way to manage the sheer volume of information.

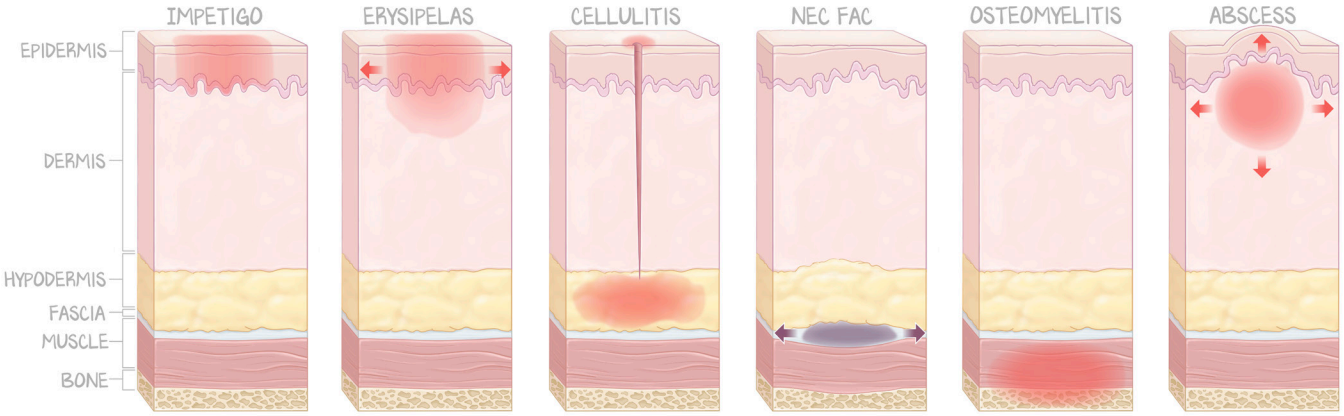

Figure 4.1: Depth of Invasion to Diagnosis
This is both an overview of the diagnoses in this lesson as well as an advanced organizer. As the illustrations moves from left to right the plane of infection gets deeper. Impetigo of the epidermis only, erysipelas of the superficial dermis, cellulitis of the hypodermis, necrotizing fasciaitis between muscle and fat, and osteomyelitis involves bone and muscle. Abscesses can be superficial or deep, from a hair follicle to erosion of muscle, and so fall outside the progression and depth model

All of the diagnoses in this lesson are infections. Because they are all infections, they all can present with sepsis—fever, leukocytosis—and they can be toxic—hypotension, lactate elevation, altered mental status. You name the diagnosis by how deep through the dermis the infection is. The organisms predict the infection's behavior—rapidly spreading outward or slowly boring downward. There is no way to link organism or depth of invasion with severity of infection. Rather than say every infection can have "fever . . . mental status" we say it once here.

How toxic a patient is, how severe the sepsis, will dictate the route of antibiotic administration (IV vs. PO) and the choice of how and how aggressively the patient is managed (outpatient vs. inpatient). These are not decisions you will need to make in the basic sciences. The goal here is to recognize the diagnosis, map it onto the depth of infection, and be able to separate the infections from one another. So rather than repeat "could have sepsis" for each diagnosis, we say it once here.

INFECTION	LAYER	BUGS	PRESENTATION
Impetigo	Epidermis only	*Strep. pyogenes* (GAS)	Tiny pustules that erupt to form honey-colored crusts
Erysipelas	Epidermis Superficial dermis	*Strep. pyogenes* (GAS or *Staph. aureus*	Extremely erythematous, raised, Sharply demarcated Acute presentation, rapid progression
Cellulitis	SubQ	*Strep. pyogenes* (GAS) or *Staph aureus*	Erythema, hot, NOT raised Obvious demarcation, but faded, not sharp Subacute presentation over days Preceded by break in skin
Nec fac Necrotizing	Fascia	*Strep pyogenes* (GAS) Anaerobes (clostridium)	Purple/black skin Erythema extends past necrosis Severe, acute, toxic Pain out of proportion with physical Crepitus (gas in tissues) Surgical emergency
Osteo	Muscle/bone	*Staph. aureus* (all) Salmonella (sickle cell) Staph epi (prosthetics) Pseudomonas (diabetes) TB (vertebral)	Subacute, insidious onset = new bone pain Hematogenous = no overt signs Direct = wounds that probe to bone Elevated ESR and CRP X-ray negative early, poorly sensitive MRI best radiographic test Bone biopsy/culture best test
Abscess Necrotizing	Any depth, locally destructive	*Staph. aureus*	Fluctuant, pus-filled mass Erythema, hot, tender over mass Spontaneous or surgical release of pus

Table 4.1: Soft Tissue Infections
A summary table that takes into account depth of invasion, organism that does the infection, and typical presentations. More information is contained in the main text.

Impetigo

Impetigo is a very superficial skin infection; it is the infection of the **epidermis only**. Neutrophils collect beneath the stratum corneum, leading to the formation of small vesicles, which eventually rupture and coalesce. Impetigo used to be caused almost exclusively by group A *Strep. pyogenes*. Now, there is an equal prevalence between group A *Strep. pyogenes* and *Staph. aureus*. You should not be asked to choose between organisms unless it is the **bullous form** of impetigo, where *Staph. aureus* is far more likely. This is because *Staph. aureus* produces exfoliatin, the exfoliative toxin, which cleaves **desmoglein** (the desmosome protein) leading to localized acantholysis.

The classic, non-bullous presentation of impetigo is flaccid pustules, almost always depicted on the face, that rupture to form a **thick honey-colored crust**. These infections are common in children. Children also are at the highest risk of complications described in the next paragraph. So almost always will a question on a licensing exam show this lesion on the face of a child.

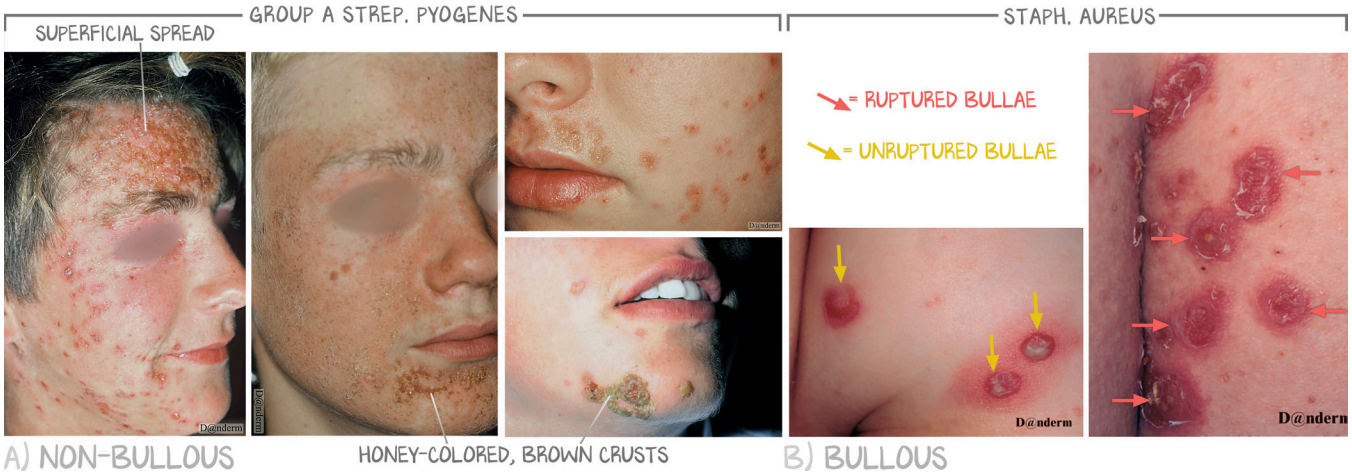

Figure 4.2: Impetigo
(a) Classically, the impetigo caused by group *A Strep. pyogenes* presents as "honey-colored" crusts on the chin. The condition may be mild and limited in location to severe and spread out. Impetigo does happen on skin regions other than the face, but it is most likely to be on a child's face on a licensing exam. (b) We want you to learn that bullous impetigo is caused by *Staph. aureus*. When you see bullae, blisters, assume *Staph. aureus*. When you see honey-colored crusts, assume Group *A Strep. pyogenes*.

A possible sequela of *Strep. pyogenes* impetigo is **poststreptococcal glomerulonephritis (PSGN)**. This is not a kidney infection, but an immune complex deposition disease. Adequate treatment of impetigo with β-lactam antibiotics (penicillin or amoxicillin) can prevent PSGN. Don't be tricked. Group A *Strep. pyogenes* can cause pharyngitis as well as impetigo, and group A strep pharyngitis can lead to PSGN or rheumatic fever. Impetigo does not cause rheumatic fever. Impetigo that is caused by *Staph. aureus* also does not cause PSGN.

Erysipelas

Erysipelas is an infection of the epidermis and **the upper dermis**. It is deeper than impetigo, but not as deep as cellulitis. It is caused by just one of the skin bugs, **group A *Strep. pyogenes.*** Because it is a strep infection and not a staph infection, erysipelas will **not be purulent or blister**. Because erysipelas is a strep infection, it will progress "*out and not down*," so will be rapidly progressive but stay in the plane of infection.

Because it is superficial, it tends to be more pronounced, more demarcated, and more acute. The affected tissue will be **red, hot, swollen,** and tender. This is an infection of the skin below the epidermis, so appears very similar to cellulitis, which is also an infection of the skin below the dermis. But because it is more superficial, the skin will have a **raised, indurated,** and **sharply-demarcated border** from the normal skin. There will be normal white skin next to extremely red, raised, and angry skin. There will be a **very acute** presentation: from normal skin to that red angry skin in a day. It typically occurs on a **unilateral leg** or the **bilateral face**. The face lesion looks like a malar rash except it **does not spare the nasolabial folds** (infections are not photosensitive).

In clinical care, the distinction between erysipelas and cellulitis is rarely made—antibiotics are given on the severity of toxicity, and those antibiotics generally cover strep and staph. In the basic sciences, distinguishing between the two is important. Focus on the acuity and the **obvious distinction** between normal and affected skin.

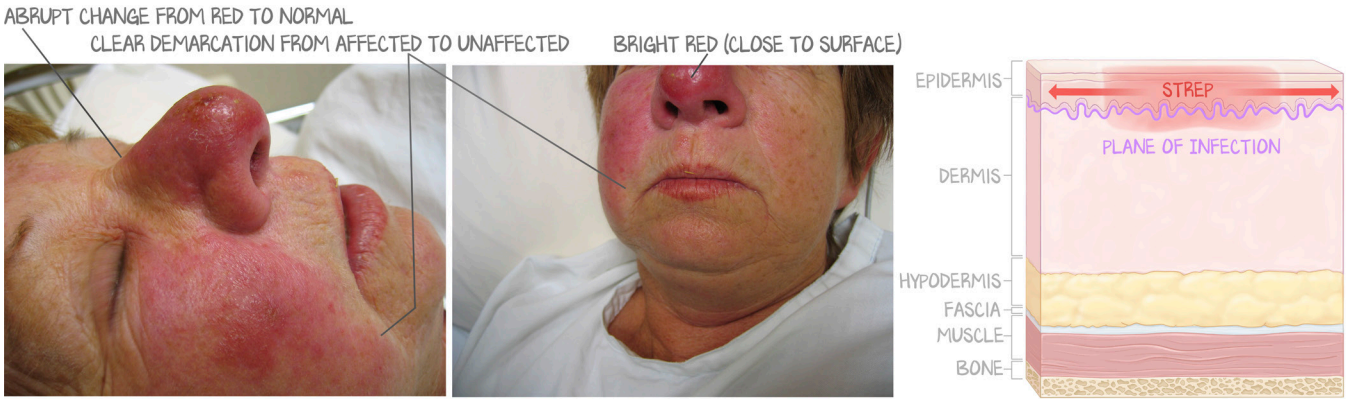

Figure 4.3: Erysipelas
This case of facial erysipelas demonstrates sharp borders between the affected (red) and unaffected (not red) skin. The margins are easy to define because the inflammation is near the surface of the skin. This can spread rapidly but usually takes a benign course. The second image demonstrates the lack of sparing of the right nasolabial fold. Because this is superficially spreading, associate it with Strep. pyogenes.

Cellulitis

Cellulitis is an infection of the **subcutaneous tissue**, deep under the epidermis. Learners must be careful to separate cellulitis from erysipelas, as they often sound very similar. Because cellulitis is deeper, it tends to be less pronounced, less demarcated, and more insidious than erysipelas.

In cellulitis, the affected tissue will be **red, hot, swollen,** and tender. Because cellulitis is an infection of the deep dermis and subcutaneous tissue, however, it will be **less demarcated**, usually will **not be raised or indurated**, and will have a more **indolent course**. The redness and heat will be faded at the edges. Students are often asked to mark the edge of the border of a cellulitis in order to track the response to antibiotics. Where erysipelas has an easily traced border, cellulitis may be more difficult—clearly defined in some skin regions, but then less obvious in others.

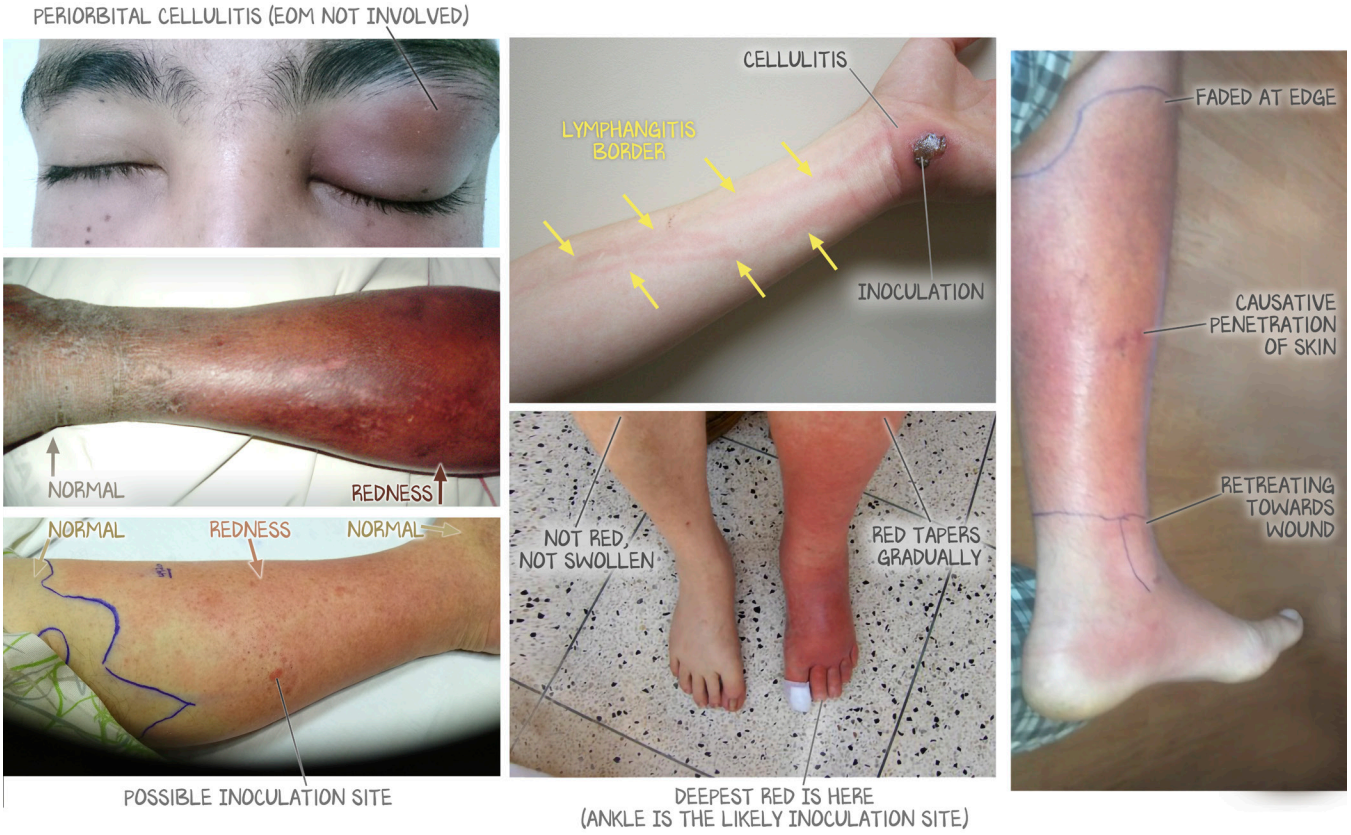

Figure 4.4: Cellulitis
Cellulitis is "subcutaneous fat-itis," an infection of the hypodermis secondary to a penetrating injury through the epidermis and dermis. Once the infection is established in the subcutaneous space, it spreads out (Group A Step. Pyogenes again). But because the infection is deep, there is no sharp demarcation between the affected and unaffected skin. There is usually an inoculation site (not visible in every photograph). The pen lines are used to assess response to therapy—the redness and warmth should retreat from the line.

Cellulitis is often caused by a **break in the skin**, a penetration of the skin barrier, inoculating the site with skin flora down to the hypodermis. Any trauma can be enough—bug bite, laceration, abrasion, puncture wound. Any vulnerability in the skin can be enough—fungal infections, furuncles. The cellulitis tends to expand **from the break in skin** outward, spreading along the hypodermis and deep dermis. The spread is slow, but persistent. When treated, the cellulitis will retreat from the edge of the cellulitis back towards the site of penetration.

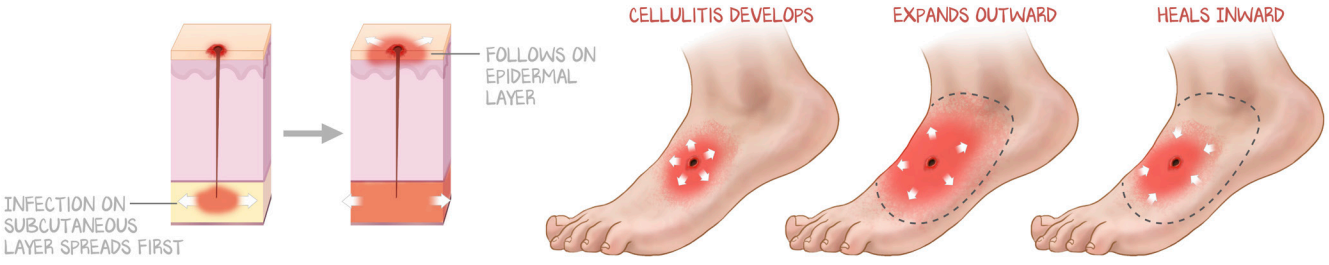

Figure 4.5: Cellulitis
Cellulitis is an infection of the subcutaneous fat, of the hypodermis. Initial innoculation allows for the infection to spread along the hypodermis. The inflammation on the surface has ill-defined borders but spreads from the site of innoculation. Treatment will show a regression of the erythema in the reverse pattern from its initial spread.

Cellulitis can be caused by many organisms, but the skin bugs—**Group A *Strep. pyogenes*** and ***Staph. aureus***—are the main offenders. Cellulitis may be purulent or nonpurulent. The presence of purulence suggests *Staph. aureus* as the causative organism. If asked to choose between them, choose group A *Strep. pyogenes* for cellulitis and *Staph. aureus* only for blistering cellulitis.

Necrotizing Fasciitis (Nec Fac)

Nec fac ("neck fack") is an infection of the **deep soft tissues** that results in **progressive destruction** of the **muscle fascia** and **overlying subcutaneous fat**. This does not get into the muscle or bone, but instead stays in the plane of the fascia. There is little resistance in this plane, which allows the infection to spread quickly. Nec fac is the "flesh-eating bacteria" that is popularized in the media. Nec fac is caused by ***Strep. pyogenes*** and not by staph. It is a quickly progressive infection (strep) and not a hearty sturdy one (staph). Nec fac can also be caused by **anaerobes** such as **clostridium**.[1]

Nec fac may start out looking like a cellulitis or erysipelas, but quickly becomes much more obvious. There will be **pain out of proportion** to the physical exam findings. The deep tissue is dying, but not the epidermis on top. Therefore, the thing you can see with your eyes (the intact skin) does not demonstrate the necrosis hidden below the dermis. You may be able to feel **crepitus** before you can see the skin changes. Crepitus, gas in the tissue, is a tell-tale sign that something is very wrong. As the infection progresses, that changes, and the skin turns to a **blue grey/purple discoloration** with or without bullae. The discoloration is not from inflammation (which is red) but instead from **necrosis** (which is black). The progression from pain-without-skin-findings to purple-skin-with-bullae is **very rapid**.

We said in the introduction that the diagnosis does not correlate to presentation. However, nec fac is the exception. Nec fac is a severe infection that causes severe inflammation. Nec fac causes necrosis of healthy tissue, which also causes inflammation. This inflammation double-whammy will present with **overwhelming sepsis** (fever, leukocytosis, tachycardia). Necrosis is tissue death, and a marker of tissue death is an elevated lactic acid. The lactic acidosis and overwhelming inflammatory response combined provoke **hemodynamic instability**. These patients are sick.

This diagnosis is a **surgical emergency**. While antibiotics and fluids will be given, **surgical intervention** is required as soon as possible. Getting ahead of the spreading infection is the only way to save the person. Getting ahead of the spreading infection often means amputation of the infected limb.

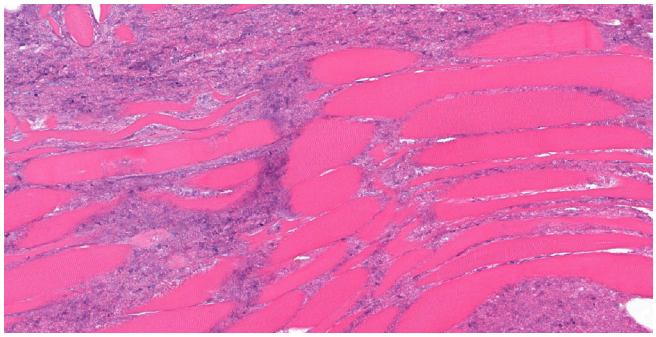

Figure 4.6: Necrotizing Fasciitis
Bacteria can quickly spread through the skin and into the fascial planes between the fat and muscle. Before signs on the skin surface are evident, much damage has already been done. The only way to get ahead of the necrosis is to operate and investigate how far the infection has spread; it will be too late if you wait for a response. Histology shows the loss of muscle (the pink stuff that looks like colloid but has striations if you get close enough) and extensive shapeless necrotic debris.

1. Clostridium species is usually associated with gas gangrene, another soft tissue infection that represents a surgical emergency and has gas in the tissues, but is not strictly necrotizing fasciitis. This becomes relevant in the clinical sciences, so is not included here.

Osteomyelitis (Osteo)

Osteo is an infection of **bone**. Bone can become infected in several ways. Bone can be infected by **hematogenous spread**, a seeding of the bone because of bacteremia. Bone can be infected by **contiguous spread** from adjacent soft tissues—the infection continues down through the epidermal layers into bone. Open **wounds** can also lead to bone infection. If the wound is through the fascia, there is no barrier to prevent microbes from entering the muscle or bone. Wound osteo is therefore usually polymicrobial. **Penetrating injuries** will directly inoculate the bone with skin flora.

Osteo is the deepest infection, and, going deep means staph. Therefore, not surprisingly, the most common cause of osteomyelitis in all comers is ***Staph. aureus***. These cases are from direct inoculation, penetrating injury, and hematogenous spread. **Salmonella** osteomyelitis is associated with sickle cell disease (with *Staph. aureus*). **Pseudomonas** (with *Staph. aureus*) osteomyelitis is associated with diabetic foot wounds and wounds penetrating through sneakers. **Tuberculosis** is associated with vertebral osteo (Pott's disease). ***Staphylococcus epidermidis*** is usually a blood culture contaminant but is also associated with **prosthetic joints** and **bone hardware** (infection of hardware is considered even deeper than osteomyelitis, prompting the removal of the hardware).

The presentation of osteo is new or worsening **bone pain**. The presentation is **subacute**, having an **insidious** onset over several days. Some osteo is easy to diagnose, as in an overlying wound where you can **probe to bone** or **see bone** with the naked eye. Some osteo has overlying cellulitis that gets better but later comes back, a product of treating the subQ infection but not the source in deep bone. Some osteo has no overt signs at all because the bone is so deep that visible signs of inflammation do not appear on the skin. Labs will show an **elevated ESR and CRP**. Though nondiagnostic, they are tracked during osteo treatment to assess efficacy of antibiotics, and having no visual identification of inflammation on the skin, are an easy lab to obtain to assess whether there is inflammation somewhere you can't see.

X-rays are specific but are insensitive early in the disease. That is, a normal X-ray does not rule out osteomyelitis, but a positive one does rule it in. Within the first two weeks, an X-ray is likely to show no changes, so **MRI** is the **best radiographic test**. A **bone culture** is the best way to confirm the diagnosis (both that the bone is infected and also by which organism it is infected).

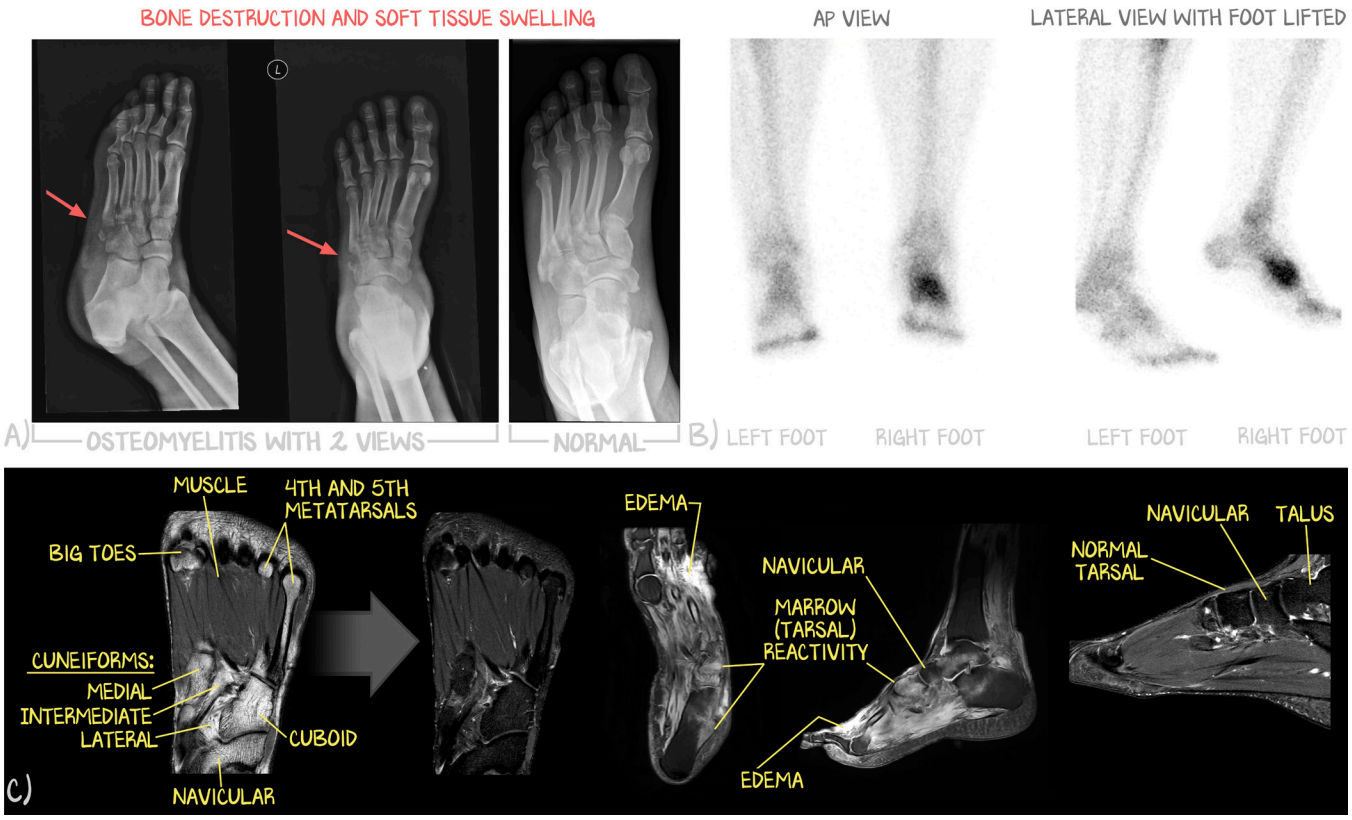

Figure 4.7: Radiographic Osteomyelitis
This figure is meant to expose you to different osteomyelitis diagnosis modalities—there's a lot here. (a) Normal foot x-ray demonstrating the bony destruction of the left fifth tarsal. X-rays diagnose obvious and ongoing osteomyelitis—if positive, it's positive; if negative, there may still be an infection of the bone. (b) Scintigraphy—a "bone scan"—is a nuclear medicine test that can detect inflammation (not infection), which is communicated by greater signal intensity, as seen in the left foot of this patient. If there is an overlying wound, this test cannot distinguish wound inflammation from bone inflammation. (c) The first (left) MRI was captured in a mode in which the bones appear white (how humans usually envision bone) to help orient you as you look at the rest of panel c. The second MRI is an unlabeled version where the bone is black. The third and fourth images are the patient's osteomyelitis. The black bone marrow is seen as white, and edema (whitest of all) surrounds the bones and foot. The fifth MRI shows you a normal comparison for the sagittal view. (d) Although MRI remains the best test, CT and 3D reconstructions are also used to diagnose osteomyelitis. More sensitive and specific than x-ray, CT is best at identifying destructive findings rather than marrow infection. (e) 3D reconstruction of the CT (created by the CT machine software) demonstrates the lytic, destructive loss of bone.

Abscess

An abscess is a collection of pus from a **walled-off infection—**full of **neutrophils and organisms**. This collection accumulates in the **dermis or subcutaneous space**. *"Strep grows out, staph grows down,"* is a phrase to remind students that cutaneous abscesses are always *Staph. aureus* and not *Staph. pyogenes*. Whereas the model we have followed has been "infection of deeper and deeper dermis," an abscess falls outside that line of thinking. Instead, think of it like a space-occupying tumor. There is **necrosis of the tissue** it occupies. The abscess eats away at the normal tissue, and leaves behind neutrophils and organisms. When drained, there may be healthy tissue missing. Abscesses can erode through the fascia into the muscle and bone.

An abscess is inflammation, so the affected tissue will be **hot, swollen, red,** and **tender**. It is an infection, so the patient may be septic—fever, leukocytosis. What defines an abscess is the mobile pus-filled pocket. This mobility we call **fluctuance**. An abscess can (and usually does) have overlying erythema, infection, or at least inflammation of the soft tissue around it. If not addressed surgically, an abscess

may have **spontaneous drainage of purulent material**. This drainage relieves the pressure and often decreases the tenderness. A skin abscess should be assumed to take care of itself—if encountered, especially if septic, an incision and drainage must be performed.

280 Test Prep. If an infection occurs in a hair follicle, a small abscess, is it called a furuncle (abscess in the hair follicle) or a carbuncle (multiple furuncles). That is to say, if the patient has the description of an abscess, but abscess is not a choice, or the abscess is being described in a hair follicle, choose furuncle or carbuncle.

Citations

Figure 4.4: Courtesy of Jerad M. Gardner, MD.